Research on Posttraumatic Stress Disorder: Epidemiology, Pathophysiology, and Assessment

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Posttraumatic stress disorder (PTSD) is a highly prevalent disorder in both clinical and community populations. This article reviews current knowledge about PTSD in order to assist clinicians in the diagnosis and treatment of reactions to traumatic life events. First, research findings are presented, followed by guidelines for the assessment of trauma and PTSD. Topics discussed include epidemiology, course, and comorbidity, as well as information processing and psychobiology. The review is limited to information about PTSD in adults, although some of the material may generalize to child and adolescent populations. © 2002 Wiley Periodicals, Inc. J Clin Psychol/In Session 58: 877–889, 2002.

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Posttraumatic stress disorder (PTSD) first appeared in the diagnostic manual of the American Psychiatric Association in 1980. The disorder, which is characterized by intrusive symptomatology, avoidance of traumatic reminders or numbing, and hyperarousal, represents an extreme and enduring reaction to psychological trauma. Before 1980, posttraumatic syndromes were recognized by a variety of names, including railway spine, shell shock, traumatic (war) neurosis, concentration-camp syndrome, and rape-trauma syndrome. The symptoms described in these syndromes overlap considerably with what we now recognize as PTSD.

In this article, we review current knowledge about PTSD in order to assist clinicians in the diagnosis and treatment of reactions to traumatic life events. First, we present research findings on epidemiology, course, and comorbidity, as well as information processing and psychobiology. With this material as background, we then discuss the assess-

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Epidemiology and Phenomenology

Prevalence

PTSD is distinguished from other psychiatric disorders in that there is a known etiological component: an event that involves life threat, serious injury, or death. Specific examples include war-zone exposure, assault, rape, torture, childhood physical and sexual abuse, natural and technological disasters, and serious accidents. Experiencing these events directly, as well as witnessing them or learning that they have happened to a loved one, can be traumatic.

The initial definition of PTSD in 1980 reflected a belief that traumatic events were rare. The current criteria no longer reflect this belief because subsequent research has shown traumatic exposure to be quite common. Using data from the National Comorbidity Survey on a large nationally representative sample of men and women, Kessler and colleagues (1995) reported that 60% of men and 50% of women have experienced a traumatic event at some point in their lives and that the majority of people who have experienced trauma report two or more events. The prevalence of trauma can be even higher in clinical populations, where 80% or more of treatment-seeking individuals may have been traumatized.

Men and women differ in the types of events they are likely to experience. The most common events overall are witnessing someone being badly injured or killed, being involved in a fire or natural disaster, and being involved in a life-threatening accident, but all of these are more common in men than in women. Men also are more likely than women to have experienced physical attack, combat, and being threatened with a weapon, held captive, or kidnapped. In contrast, women are more likely to have experienced rape, sexual molestation, childhood parental neglect, and childhood physical abuse.

General population estimates of lifetime PTSD in the National Comorbidity Survey are 5% for men and 10% for women. Among traumatized individuals, the lifetime prevalence of PTSD is 8% in men and 20% in women. The prevalence of PTSD in treatment-seeking populations is much higher, sometimes reported to be in excess of 50% even among individuals who are not seeking specialized trauma care.

The likelihood of developing PTSD varies with the type of trauma experienced. Such differences between traumas provide a possible explanation for the gender differences in PTSD prevalence—namely, that women are more likely to develop PTSD because of the nature of the traumas they tend to experience. For example, rape, which has the highest likelihood of resulting in PTSD, is much more likely to happen to women (9%) than to men (<1%). However, gender differences in exposure do not explain completely gender differences in prevalence. Women remain four times more likely than men to develop PTSD even when type of trauma is taken into account (i.e., controlled for in statistical analysis). The gender difference in PTSD prevalence requires further explanation, but has important clinical implications nonetheless.

The likelihood of developing PTSD varies with other risk factors, including both situational and personal characteristics. Findings on race have been inconsistent, with some studies showing nonwhites to be at higher risk of PTSD than whites, even when the prevalence of traumatic exposure is taken into account. Younger age and lower education are associated with increased risk. Increased risk of PTSD also is associated with preexisting psychiatric disorder, early conduct problems, childhood adversity (e.g., parental

loss, economic deprivation), personality pathology (especially neuroticism), family history of psychiatric disorder, severity of initial reaction, peritraumatic dissociation (i.e., during and shortly following a trauma), and poor social support after a trauma.

The severity of a trauma also is an important risk factor. Trauma severity refers to such features as the duration of a trauma, the number of times it occurred, and the extent of injury or life threat. The magnitude of the relationship between trauma severity and PTSD is only moderate, but it is highly consistent across populations and trauma types: the greater the trauma severity, the greater the likelihood of PTSD or severity of PTSD symptoms.

Posttraumatic reactions fall on a continuum, and many clinicians and researchers believe it is useful to conceptualize partial syndromes as well. Definitions of partial PTSD typically require the exposure ("A"), impairment ("E"), and duration ("F") components of a diagnosis, and some combination of symptoms. Usually, the "B" (intrusion) symptom criterion must be met along with some combination of the "C" (avoidance–numbing) and "D" (hyperarousal) criteria.

Course

On average, posttraumatic symptoms decrease over time, but the course of PTSD varies both within and between people. DSM-IV includes several specifiers for providing information about the course of PTSD. An *acute* case is one in which the duration of symptoms is less than 3 months. A *chronic* case is one in which symptoms last 3 months or longer. *Delayed onset* of PTSD is diagnosed when at least 6 months have elapsed between traumatic exposure and the onset of symptoms.

Most cases of PTSD are not acute, as defined in DSM-IV. Roughly 90% of PTSD cases in the National Comorbidity Survey had a duration longer than 3 months, and over 70% had a duration longer than a year. More than one-third of cases never recovered. The median time to remission (the time by which 50% of cases recovered) was 36 months among individuals who received treatment and 64 months among individuals who did not. Although these findings do not definitively show that treatment shortens the duration of PTSD, they at least identify the absence of treatment as a risk factor for chronicity. Other risk factors for chronicity include female gender and a history of psychiatric disorder. Research in this area is emerging, and it is not possible to say whether the risk factors for the development of PTSD are the same as the risk factors for maintenance.

The exact percentages of people who have immediate versus delayed onset, a single versus multiple symptomatic episodes, and a variable versus a stable course are unknown. However, delayed onset does not appear to be the norm, nor does a stable course. Individuals who are asymptomatic at one time may find that stressors reactivate their symptoms, both related and unrelated to the initial trauma. Reactivation sometimes occurs in people who have been asymptomatic for years. Typically, such cases are not extreme examples of delayed onset, but rather are examples of symptom recurrence or delayed recognition.

Acute Stress Disorder

DSM-IV introduced the diagnostic category of acute stress disorder (ASD) for severe reactions to traumatic stressors during the month following a traumatic event, before a diagnosis of PTSD can be made. The symptom criteria for ASD include three or more dissociative symptoms, one or more intrusive symptom(s), marked avoidance, and hyper-

arousal. The symptoms must cause clinically significant distress or impairment, last between 2 days and 4 weeks, and occur within 4 weeks of the traumatic event.

The diagnosis of ASD has been controversial since its introduction (Bryant & Harvey, 2000). One concern was a lack of information about ASD. Another concern was that the diagnosis pathologized normal reactions to traumatic events, which can be extreme for many individuals who do not develop PTSD. Both of these concerns have been addressed by research that has emerged since 1994. Estimates of the prevalence of ASD following trauma range considerably, from less than 10% to over 30%. Much attention has focused on the utility of ASD in predicting the development of PTSD and on the adequacy of the diagnostic criteria. Whereas ASD is highly predictive of PTSD, the inclusion of dissociative symptoms in the diagnostic criteria limits the predictive utility of the diagnosis. Individuals who do not have the necessary dissociative symptoms but who otherwise meet ASD criteria also are at increased risk of developing PTSD, relative to individuals who do not meet the other ASD criteria. In general, these findings are consistent with data from prospective studies that have found the intensity of an initial reaction to a traumatic event to predict the subsequent development of PTSD.

Comorbidity

Chronic PTSD often is accompanied by other Axis I psychiatric disorders and general impairment of psychosocial function. In the National Comorbidity Survey, Kessler and colleagues (1995) reported that 88% of men and 79% of women with lifetime PTSD had at least one comorbid diagnosis. Major depression was the most common comorbid diagnosis, occurring in just under half of men and women with PTSD. PTSD was associated with higher prevalence of all disorders studied: major depression, dysthymia, mania, generalized anxiety disorder, panic disorder, simple phobia, social phobia, agoraphobia, alcohol abuse/dependence, drug abuse/dependence, and conduct disorder. Although a history of psychiatric disorder is a risk factor for developing PTSD following a traumatic event, PTSD often leads to the development of psychiatric disorder as well. In the National Comorbidity Survey, PTSD was primary for the majority of cases in the development of affective and substance-use disorders, and also in the development of conduct disorder among women.

Symptom overlap may explain at least part of the high lifetime comorbidity seen in PTSD. For example, both PTSD and major depression share sleep disturbance, impaired concentration, and diminished interest in one's surroundings. In addition, emotional detachment and restricted range of affect in PTSD may be confused with depressed mood and psychomotor retardation in major depression. Symptoms found in both PTSD and generalized anxiety disorder include autonomic hyperarousal, irritability, hypervigilance, exaggerated startle, impaired concentration, and insomnia. In addition, PTSD and panic disorder both exhibit autonomic hyperarousal and dissociation, but, perhaps more important, the psychological and physiological reactivity in PTSD sometimes can appear to be a full-fledged panic attack.

The prominence of comorbidity and symptom overlap between PTSD and other disorders raises important theoretical and clinical issues. Theoretically, one may ask whether a person who meets diagnostic criteria for PTSD and major depression has two distinct disorders or a depressed subtype of PTSD. Neurobiological research on hypothalamic-pituitary-adrenocortical function in both disorders has prompted some investigators to propose that comorbid PTSD and major depression is much more similar to PTSD alone

and distinctly different from major depression alone (Yehuda & McFarlane, 1997). Nevertheless, the symptoms of comorbid disorder require clinical attention, regardless of their origin.

Clinically, comorbidity and symptom overlap demand that all new psychiatric patients receive a thorough diagnostic assessment, including a comprehensive trauma history. Unfortunately, this is not always the case. As a result, a patient with severe PTSD who presents as an acute emergency with suicidal behavior or autonomic hyperarousal or alcohol/drug intoxication may be diagnosed and treated exclusively for major depression or panic disorder or substance abuse. It is not unusual to discover afterwards that the acute psychiatric emergency was due primarily to the underlying PTSD. Failure to conduct a comprehensive assessment in such cases may result in poor clinical management, and sometimes in tragic consequences.

It is important to consider medical as well as psychiatric comorbidity among individuals with PTSD. There is mounting evidence that traumatic exposure increases the risk of poor physical health (Schnurr & Jankowski, 1999). Trauma survivors, relative to nontraumatized individuals, report more medical symptoms, use more medical services, have more medical illnesses confirmed by a physician's examination, and display higher mortality. A few studies have suggested that PTSD plays an important mediational role in the relationship between trauma and physical health. This makes sense because numerous correlates of PTSD—neurobiological (including immunological) abnormalities and psychological factors (depression, hostility, negative health behaviors, and poor coping abilities)—are associated with increased risk of medical illness. One implication of the findings on trauma and physical health is that primary and specialty medical practitioners should screen for trauma and PTSD, and mental-health providers should attend to the psychological aspects of physical-health problems in their traumatized patients.

Complex PTSD

Some clinicians and researchers have argued that the PTSD diagnostic criteria do not capture adequately clinically significant symptoms exhibited by people who have been exposed to protracted interpersonal trauma such as childhood sexual abuse, domestic violence, and torture. The symptoms, known collectively as "complex PTSD" or "DES" (Disorders of Extreme Stress), entail impulsivity, problems with affect regulation, dissociative symptoms, self-destructive behavior, abnormalities in sexual expression, and somatic symptoms.

Complex PTSD rarely occurs without PTSD. In a study of DSM-IV Field Trial participants who reported sexual or physical abuse, 2% of the sample had DES only, 19% had PTSD only, 48% had both PTSD and DES, and 30% had neither (Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997). And although theoretically predicted to occur in survivors of a range of interpersonal traumas, complex PTSD is associated most strongly with sexual abuse. In the Roth et al. study, complex PTSD occurred in 27% of participants with physical abuse only, 54% of participants with sexual abuse only, and 79% of participants with combined physical and sexual abuse.

Complex PTSD is mentioned in DSM-IV as an associated feature of PTSD, and there has been debate about whether it should be included as a separate diagnosis. There is general agreement, however, that patients who exhibit this pattern of symptoms often present a very difficult clinical challenge. Many clinicians find the concept of complex PTSD to be useful for understanding the needs of these patients.

Ethnocultural Considerations

The epidemiological and comorbidity findings presented above come from studies of American men and women. PTSD prevalence is higher where exposure to war, state-sponsored terrorism, or interpersonal violence is more common than in the U.S. A recent epidemiological study estimated that the prevalence of lifetime PTSD is 37% in Algeria, 28% in Cambodia, 16% in Ethiopia, and 18% in Gaza (de Jong et al., 2001).

The diagnostic criteria for PTSD and most of the current data pertaining to this disorder are based primarily on research and clinical experience with North American or European individuals. Indeed, PTSD has been criticized from a cross-cultural perspective because of this Western, industrialized, Euro-American bias. The question is not whether PTSD can be detected among traumatized people from traditional ethnocultural settings—it can. Rather, the question is whether there are other distinctive ethnocultural posttraumatic idioms of distress that fall outside strict DSM-IV guidelines. It is important to understand how traumatic exposure is perceived and expressed in different ethnocultural settings because different clinical interventions may be required than those that have proven effective for Euro-American PTSD patients.

Information Processing in PTSD

Memory and concentration difficulties are frequent complaints among people who have PTSD. Findings regarding the exact nature of cognitive problems in PTSD are inconsistent. For example, some data show the deficits to be fairly specific, whereas other data show the deficits to be more general. However, one point is clear: the subjective complaints of cognitive problems are corroborated by poor performance in assessments of cognitive function. An exciting area of PTSD research is focused on answering questions about the extent to which these cognitive problems in PTSD are related to alterations of brain structures involved in learning and memory.

Several authors have proposed information-processing models of PTSD. Foa and her colleagues have offered a model based upon the concept of a *fear structure*, which they describe as "a network in memory that includes three types of information: information about the feared stimulus situation; information about verbal, physiological, and overt behavioral responses; and interpretive information about the meaning of the stimulus and response elements of the structure" (Foa & Rothbaum, 1997, p. 74). Fear structures are programs that enable individuals to escape or avoid a feared stimulus. They differ from other information structures in memory not only because of their particular stimulus and response components, but also because fear structures contain information about danger. Foa and her colleagues have proposed that treatment must be based upon the activation and correction of information in fear structures, which is accomplished by exposure to traumatic stimuli and cognitive restructuring, respectively. The model has yielded much productive research and has served as the basis for a theoretically grounded approach to treatment (see Jaycox, Zoellner, & Foa, this issue).

Ehlers and Clark (2000) recently proposed a cognitive theory in which PTSD persists only if an individual processes a traumatic event in a way that causes the event to threaten the individual after it has occurred. The perception of current threat leads to intrusions and other re-experiencing symptoms, arousal, anxiety, and distress. The perception of threat also leads to behavioral and cognitive attempts to reduce the immediate sense of threat that actually serve to maintain symptoms. For example, thought suppression, used to avoid unwanted memories, may increase intrusive recollections. Selective attention to threat cues also may increase intrusions and distress. The implications of this model for

treatment are that trauma memories need to be integrated into a patient's experience, and dysfunctional appraisals and maladaptive behavioral and cognitive strategies need to be altered.

Psychobiology of PTSD

There is a rich tradition of research on stress, coping, and adaptation that serves as a useful context in which to understand many of the psychobiological abnormalities detected among individuals who have PTSD. A number of key biobehavioral systems have been selected through evolution to promote survival of the human species. These include: sympathetic nervous system (SNS) activation; mobilization of the hypothalamic-pituitary-adrenocortical (HPA) axis; fear conditioning; and the startle reflex. We summarize below how these systems are affected by PTSD, using material from several reviews (Friedman, 1999; Friedman, Charney, & Deutch, 1995; Yehuda & McFarlane, 1997).

Activation of SNS and central adrenergic mechanisms is a key component of the human stress response. Normally, when the crisis has passed and coping has been successful, adrenergic activity returns to pre-crisis normal homeostatic levels. That is not so for people with PTSD, who exhibit continuously elevated SNS and adrenergic function, even in the absence of an external threat. For example, people who have PTSD exhibit SNS hyperresponsivity to a variety of neutral and trauma-related stimuli presented in a laboratory. Elevated concentrations of adrenergic metabolites (catecholamines) are found in their urine. Some (α-2 and β) adrenergic receptors are downregulated and are unusually sensitive to certain drugs (e.g., yohimbine) that disinhibit the CNS adrenergic system. In short, the adrenergic system in people who have PTSD appears to have been recalibrated to deal with a permanent life-threatening crisis.

The HPA system is the other key biobehavioral system involved in the human response to stress. Here, too, there is strong evidence showing dysregulation of HPA function among individuals with PTSD, although results are mixed concerning the precise abnormality detected in different experiments. Elevated levels and activity of corticotropin-releasing factor have been shown. A number of studies have found increased lymphocyte glucocorticoid receptor levels and supersuppression to the glucocorticoid dexamethasone during the dexamethasone suppression test. More variable findings have emerged from studies that measured 24-h urinary cortisol, with some results (mostly with male subjects and post-menopausal women) showing reduced levels, and other results (mostly with premenopausal women) showing elevated levels. Furthermore, urinary cortisol appears to vary according to the degree of psychological stress to which subjects are exposed at the time it is measured.

Fear conditioning is an adaptive mechanism whereby animals learn to preserve information about previous threats in order to promote future survival. The most elegant research demonstrating the presence of fear conditioning among individuals with PTSD involves laboratory paradigms in which these individuals are exposed to auditory or visual stimuli pertaining to their traumatic event. For rape victims, this might be stimuli about sexual assault. For military veterans, it might include war-zone-related stimuli. For both, it might include a brief audiotaped autobiographical synopsis of their specific traumatic episode. Most people who have PTSD will exhibit sudden dramatic elevations of cardiovascular or other SNS activity immediately after exposure to such trauma-related stimuli in a laboratory setting.

The startle response reflects the organism's alarm system upon exposure to threatening stimuli. Abnormal findings in startle-reflex research with people who have PTSD

include shorter latency and increased amplitude, resistance to normal habituation, and loss of the normal inhibitory modulation of the startle reflex.

Well-established abnormalities detected in other major biobehavioral systems among individuals with PTSD include alterations in the diurnal sleep cycle, elevated thyroid function, and dysregulation of the opioid system. Less well-established, but strongly supported by relevant animal or human research, is the likelihood that PTSD may be associated with suppressed immunologic function, kindling/behavioral sensitization of specific brain nuclei (especially in the hippocampus and other limbic system sites), abnormal serotonergic activity, and altered glutamatergic (excitatory amino acid) mechanisms that adversely might affect information processing and memory function.

Given the wide spectrum of psychobiological abnormalities detected among individuals with PTSD, it might be expected that they also would exhibit abnormalities in brain structure and function. Although research on this possibility is in its infancy, several independent laboratories using magnetic-resonance imaging have shown reduced hippocampal volume among males and females with PTSD who have been exposed to sexual, war-zone, and motor-vehicle trauma, respectively. Functional brain abnormalities are suggested by two investigations with positron-emission tomography in which individuals with PTSD, exposed to trauma-related stimuli, exhibited increased regional cerebral blood flow to brain structures (e.g., limbic and paralimbic areas, especially the amygdala and anterior cingulate gyrus) that appear to play key roles in processing emotional information.

Assessment and Diagnosis

Assessment and diagnosis provide a platform for case formulation, treatment planning, and outcome monitoring. We encourage readers to examine the volume by Wilson and Keane (1996) for more detail about instruments, as well as a comprehensive discussion of many broader issues.

Assessment of Trauma

The first step in the diagnosis of PTSD is determining whether an individual has been exposed to a traumatic event, or, as often will be the case, determining to which events an individual has been exposed. This step is important because exposure is one of the diagnostic criteria for PTSD and because all of the "B" (re-experiencing) symptoms and two "C" symptoms must be evaluated with respect to a specific event or events. The decision to ask about a single event or to obtain a lifetime trauma history (and then perhaps focus on a few key events) should depend on whether one needs to know if an individual has PTSD in response to a specific event only, that is, whether a childhood sexual-trauma survivor who has been in a motor-vehicle accident meets criteria for PTSD related to the accident. If not, PTSD may be assessed in response to a worst event or a constellation of a few worst events.

The first diagnostic criterion for PTSD, as listed in DSM-IV, requires that an individual has been exposed to (or witnessed or has been confronted with) a life-threatening event to which the individual responded with "fear, helplessness or horror." Clinical judgment may be required to determine whether an event was life threatening. Individuals sometimes exaggerate, but also sometimes minimize, the life threat in a given situation. Assessing whether serious injury or death occurred for someone involved in the situation can be very useful for ascertaining life threat, although many traumatic events do not result in actual injury or death to anyone. Some suggested probes are:

> Were you or others in the situation seriously injured or harmed? [If NO, or NOT SURE] Did you think that your life was in danger or that you might be seriously injured? Did you think that others might be killed or seriously injured? Under such circumstances, do you think that other people would feel that their lives were in danger or that they might be seriously injured?

Clinical judgment also is required to determine an individual's response to a trauma. Again, exaggeration may be a problem, but it is not uncommon for people to report being numb or "in shock." In this situation, asking about an individual's reaction at some point after the trauma—"when the shock wore off"—can be useful.

Our mention of the importance of clinical judgment raises the question of whether it is better to use interviews or questionnaires for assessing trauma. There is no absolute answer to this question. Obviously, interview assessments can be essential for dealing with exaggeration and minimization, but interviews may be subject to problems of interrater reliability, as well as the unwillingness of some people to disclose to another person what they might disclose in an anonymous questionnaire. Questionnaires can provide a more comfortable format than interviews for people to relate distressing experiences, but care must be taken during the administration of questionnaires to ensure that appropriate clinical contact is available if needed.

The small amount of information on the performance of questionnaire versus interview assessment suggests that questionnaires slightly underestimate amount of exposure. A possible reason for the difference is that interviews provide opportunities for clarification about question wording, whereas questionnaires typically do not. This can be especially important in the assessment of sexual trauma because individuals may be unsure about categorizing some sexual experiences they have had, for example, date rape. One practice commonly employed in many trauma questionnaires and interviews is for questions to be behaviorally specific, that is, defined in terms of the behaviors involved ("inserted a penis, finger, or other object into your vagina") rather than summary labels (e.g., "vaginal rape").

Reports of event exposure are reasonably, but not perfectly, consistent from one occasion to another. Research has not determined conclusively why individuals may report an event on one occasion and fail to report it on another. Simple forgetting is a likely explanation for some inconsistency, as is reluctance to retell a given event to a given person on a given day. There is a widely held clinical impression that either traumatic exposure or PTSD itself may interfere with the retrieval of traumatic memories in some individuals. This issue currently is generating important research on the nature of memory processes in general and traumatic memories in particular.

A related issue is the validity of a report of trauma on a single occasion. During the 1990s, the question of how to determine the validity of a traumatic memory provoked a great deal of controversy because of concerns that memories of traumatic events can be implanted falsely by inappropriate clinical practices. Thorough discussion of the issue is beyond the scope of this article, but we note that there is general consensus that suggestive memory-recovery techniques should be used with extreme caution in clinical interactions with patients who have unclear memories, or no memories at all (Wilson & Keane, 1996).

Some researchers have attempted to examine the validity of individuals' reports of exposure to traumatic events by comparing these reports with archival sources such as combat medals or state records of child abuse. The comparisons have shown that self-reports and archival records are correlated reasonably, but that individuals may sometimes fail to report documented trauma. An important problem with archival sources is that they are not a complete record of an individual's experiences and may underestimate

exposure. Documented occurrences may be very good indicators of exposure, but lack of documentation does not necessarily indicate that exposure never took place. It is helpful to seek out archival and collateral sources of information when possible (and appropriate), but these sources should be used in conjunction with an individual's own report to develop a comprehensive picture of his or her trauma history.

Assessment of PTSD

The current diagnostic criteria for PTSD categorize symptoms into three clusters. The first cluster consists of symptoms of persistent re-experiencing of the traumatic event; the second, avoidance of stimuli associated with a traumatic event or numbing of general responsiveness (not present before the event); and the third, increased arousal. These symptoms must last for at least one month and must result in either significant distress or psychosocial impairment. The criteria present a challenge for both the traumatized individual and the assessor to determine explicitly to what extent the individual's symptoms are a *reaction* to a trauma. This task is straightforward for re-experiencing and avoidance symptoms, which are referenced literally to the trauma, but less so for numbing and hyperarousal. For these symptoms, it is necessary to determine whether their onset occurred after the trauma, or if not, whether their severity intensified after the trauma. It may not be possible to ascertain the temporal relationships between trauma exposure and trauma-nonspecific symptoms in an individual who is traumatized at a young age. In such cases, the symptom may be considered to be trauma related.

At times, obtaining a lifetime trauma history can lead to complications for the clinician who, upon determining that a traumatized individual has experienced more than one event, must decide which event, or events, to use as the basis for PTSD assessment. It is uncommon to assess whether an individual has PTSD in response to every event that has been experienced. A more typical practice is to assess PTSD with respect to a "worst" event, a first event, a most recent event, or some manageable combination of events simultaneously. The latter particularly can be useful because individuals who have experienced multiple traumas often will have a blended mapping of traumas to symptoms, such as, nightmares about sexual abuse but intrusive recollections about both the abuse and a serious auto accident.

Questionnaire measures of PTSD fall into three general categories: those consisting only of items that represent the symptoms needed for diagnosis; those consisting of items needed for diagnosis, as well as associated features such as guilt; and those developed empirically from items taken from general-symptom inventories. The first two types of questionnaires can be used to determine whether an individual has the sufficient number and type of symptoms needed for diagnosis. All questionnaires can be used to determine whether an individual is likely to have a diagnosis based on an overall severity score. For some questionnaires in particular, this severity-threshold approach is a highly valid way to estimate a diagnosis. Besides their obvious value in assessing a large number of people in a cost-efficient manner, questionnaires are useful for screening and for tracking symptom severity.

However, structured interview measures of PTSD typically are considered to be the gold standard for diagnosis. Although clinical interviews often are used by busy practitioners, the additional time that can be required to administer a more structured interview is justified by the completeness and accuracy such methods foster. Available interviews vary in terms of whether they require a clinician or a layperson for administration, provide severity information along with a diagnosis, and include questions about traumatic

exposure and associated symptom features. Layperson-administered interviews typically are used in research settings, either for screening or when budget constraints preclude the participation of clinicians. These interviews can have acceptable to very good validity when lay interviewers use instruments designed for non-clinicians and are trained carefully and monitored.

There are a number of excellent structured interviews available for clinically trained researchers and practitioners. Here, we describe in detail one of them, the Clinician-Administered PTSD Scale, or CAPS (Weathers, Keane, & Davidson, 2001). The CAPS was developed as an interview for diagnosing current and lifetime PTSD and for quantifying PTSD symptom severity. It can be used to assess current and lifetime status, as well as weekly change. The CAPS consists of questions about the 17 PTSD symptoms listed in DSM-IV, duration, course, impairment, and associated symptoms, including guilt, suicidality, and dissociation. It also includes a simple and clinically helpful trauma assessment for determining the event(s) to be used as the basis for PTSD assessment.

Several aspects of the CAPS help to make it an informative and easy-to-administer interview. For each symptom, respondents are asked about the symptom's frequency, which is rated on a 5-point scale. Then, provided that the symptom occurs, respondents are asked about its intensity, which also is rated on a 5-point scale. Ratings of frequency and intensity are correlated highly. However, distinguishing them can be important for getting a good picture of intense but low-frequency events, as well as for tracking symptom change, which may occur by decreases in frequency or intensity, but not both.

A sample CAPS item is illustrated in Figure 1. The figure highlights another useful feature of the CAPS: behavioral descriptions to help the interviewer distinguish between each point on the frequency and intensity scales. A "QV," or "questionable validity," specifier is included for indicating concerns about a rating's quality, such as vague or inconsistent examples or poor comprehension on the part of a respondent. A "Trauma-

2. (B-2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.

Frequency	***Intensity***	Past week
Have you ever had unpleasant dreams about (EVENT)? Describe a typical dream. *(What happens in them?)* **How often have you had these dreams in the past month** *(week)*?	**How much distress or discomfort did these dreams cause you? Did they ever wake you up?** [IF YES:] *(What happened when you woke up? How long did it take you to get back to sleep?)* [LISTEN FOR REPORT OF ANXIOUS AROUSAL, YELLING, ACTING OUT THE NIGHTMARE] *(Did your dreams ever affect anyone else? How so?)*	F ____ I ____ Past month F ____ I ____ Sx: Y/N Lifetime F ____ I ____ Sx: Y/N
0 Never 1 Once or twice 2 Once or twice a week 3 Several times a week 4 Daily or almost every day	0 None 1 Mild, minimal distress, may not have awoken 2 Moderate, awoke in distress but readily returned to sleep 3 Severe, considerable distress, difficulty returning to sleep 4 Extreme, incapacitating distress, did not return to sleep	
Description/Examples	***QV (specify)*** _____	

Figure 1. Question example from the Clinician-Administered PTSD Scale.

related" specifier additionally is included on items that are not referenced specifically to the trauma, for example, restricted range of affect. This specifier permits an interviewer to acknowledge a symptom's presence while discounting it for diagnostic purposes.

The severity of an individual symptom is computed by summing intensity and frequency ratings. A total severity score is computed by then summing the individual symptom-severity ratings. The presence or absence of each symptom may be determined according to several rules. The most widely used rule is to count a symptom as present if it is at least a "1" in frequency and a "2" in intensity, that is, present and of at least moderate intensity. Research indicates that this rule probably is too liberal, leading to the overdiagnosis of PTSD. However, the "1–2" rule is useful for clinical purposes. A more stringent scoring criterion, such as the "1–2" rule in combination with a minimum severity of 65, should be considered when diagnostic validity is an important concern, such as in a forensic context.

Multimodal assessment of PTSD is recommended. Combining questionnaire and interview data, and, if possible, psychophysiological and neurobiological data, provides the most comprehensive means of making a diagnosis and characterizing an individual's posttraumatic reactions and clinical needs. Unless you only wish to determine whether a given individual has PTSD, it is desirable to assess other symptoms, as well as psychosocial impairment. As noted in our discussion of comorbidity, chronic PTSD often is associated with a complex array of problems that can affect many domains of functioning.

Concluding Remarks

PTSD is a disorder with a presumed etiology—a traumatic event. Yet most people who experience a trauma never develop PTSD. The relatively low prevalence of PTSD among individuals who are traumatized raises the question of what factors besides a trauma are required in order for PTSD to develop. Studies published in the early and mid-1980s tended to show little, if any, contribution of premorbid characteristics, such as childhood environment. After almost two decades of study, a large amount of data suggests that the etiology of PTSD is understood best in terms of what an individual brings to a traumatic event, as well as what he or she experiences afterward, and not just characteristics of the event itself. A valuable result of this research has been an increased understanding of the role of risk factors in the etiology of trauma exposure itself. Recent prospective studies of acute-trauma survivors have helped us understand much more about the development of PTSD, and ultimately may help us accurately identify at-risk individuals in order to prevent PTSD from occurring.

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